Advances in Biological Research 9 (4): 247-252, 2015
ISSN 1992-0067

DOI: 10.5829/idosi.abr.2015.9.9559

Large Strongyle Parastes in Equine: A Review

Anmaw Shite, Bemrew Admassu and Alimaw Abere

University of Gondar, Faculty of Veterinary Medicine,
Department of Veterinary Pharmacy and Biomedical Sciences, P.O. Box: 196, Gondar, Ethiopia

Abstract: Strongylosis is one of the most important internal parasitic diseases of equines caused by nematodes of strongylidae family affecting more than 80% equids in the world. Strongylus vulgaris (S. vulgaris) is one of the large strongyle and the most prevalent and pathogenic parasites of equines. Large strongyles show major pathogenesis that is encompasses verminous arteritis, damage of visceral organs, embolism or thrombosis leading to death and is mainly attributed to migrating larvae of parasites. The larvae of *Strongylus species* are causes to large nodules in the wall of caecum and colon with a considerable hemorrhage and the nodules become rupture and release the worm into the lumen of the intestine. In the heavy burdens bleeding can occur and leads to the death of the animals. Inspite of substantial improvements in understanding the life cycle of strongyles and adopting latest diagnostic techniques and implementing the most modern treatment and control measures, the disease is still prevalent and could not be eradicated from any part of the world. The current strategy engaged in seasonal use of anthelmintics is the key to arrest the disease and overcome anthelmintic resistance. Using a mixed grazing system and removal of all horse feces from the fields twice weekly is highly effective for the control of strongyles.

Key words: Strongylosis · Equines · *S. vulgaris* · Migrating Larvae

INTRODUCTION

The equine population in Africa is 17.6 million of which 11.6 million, 2.3 million and 3.7 million donkey, mules and horses, respectively. Ethiopian retains a total of 8.6 million equines, 5.2, 2.8 and 0.6 million donkeys, horses and mules, respectively. This high number of equine in the production system shows the importance of equine species in the area [1]. Most equines are found in the area of high human population density where the production system is dominated by annual cropping with livestock production [2].

The large strongyles are nematode parasites which are classified under the family *strongylidae*. These parasites live in the large intestine specifically caecum and colon of equines. The three most important species of large strongyles found in equines are *S. vulgaris*, *strongylus edentates* (*S. edentates)* and *strongylus equines (S. equines)* [3]. The common name of *S. vulgaris* is called as double tooth strongyle*, S. edentatus* is called as toothless strongyle and *S. equinus* is called as tripled toothed strongyle. *S. vulgaris* is smaller than the other two large strongyle species [4].

Strongylosis has been reported from all parts of the world and almost affects more than 90% of horse population [5]. In Ethiopian studies, *S. vulgaris* was very common and highest in equids in mid and high altitude areas where the rain fall is relatively high and follows as a bimodal pattern [6]. Among the gastro-intestinal nematodes of horses, large strongyle infections, *S. vulgaris* has long been considered as one of the most common and pathogenic parasites of the horse [7, 8]

The description of very complex life cycle of the strongylids of the large strongyles was initiated by Wetzel in 1941 and then described more precisely by Enigk ten years later in 1951. Further studies conducted by McGraw and Slocombe in 1977 [9]. When the equines ingest the larvae; it travels through the digestive system to the large intestine. *S. vulgaris* migrate to the anterior mesenteric artery; *S. edentatus* to the liver (flank area) and *S. equinus* migrate to the liver and pancreas [10]. Adult large strongyles live in the caecum and colon.

Corresponding Author: Anmaw Shite, University of Gondar, Faculty of Veterinary Medicine,
Department of Veterinary Pharmacy and Biomedical Sciences, P.O. Box: 196, Gondar, Ethiopia.

Fourth (L4) and fifth (L5) stage larvae are responsible for arteritis, necrosis and fibrosis of the cranial mesenteric artery and its branches [11, 12]. Severe colic and death of horses is the consequence of thrombosis and embolism leading to infarction of the intestinal tract [13].

The diagnosis of large strongyle is by examination of feces for the detection of the strongyle egg, fecal culture for identification of strongyle larvae and per rectal examination reveals aneurysm of cranial mesenteric artery [4]. Equines are treated with anthelmintic drugs to eliminate adult strongyles from the large intestines and to prevent excessive contamination of pastures with eggs and L3s [14]. The effective control programs of the large strongyles are applying a strategic treatment and improve pasture management [15].

Therefore, the Objectives of this Paper Are:

- To review the characteristics of large strongyle.
- To highlight the epidemiology, life cycle, pathogenesis, clinical sign To indicate the possible treatment and control options.

General Characteristics and Morphology: Large strongyles are classified under the kingdom animalia, phylum *nemathelminthes*, class *nematoda,* order strongylida, superfamily *strongyloidae,* family *strongylidae* and subfamily *strongylinae*. The sex of large strongyles is separate that is female and male. Females are oviparous and eggs are strongyle type. They are reproduced by sexually and they do not have circulatory system (no blood vessel) and respiratory system. Grossly these parasites look robust dark red worms which are easily seen against the intestinal mucosa. They have cylindrical unsegmented shape, body cavity, intestine with anus, nervous system and some has prominent leaf crown surrounding the mouth opening [4].

The well-developed buccal capsule of the adult parasite is prominent as the bursa of the male. Microscopically species differentiation is based on size, presence or absence of teeth and the shape of the teeth in the base of buccal capsule. The *S. vulgaris* has 1.5-2.5cm long and two ear-shaped rounded teeth; the *S. edentatus* has 2.5-4.5cm long, with no teeth; *S. equinus* has 2.5-5cm long with three conical teeth and one is situated dorsally and larger than others [3]. The shape of the buccal capsule in each large strongyle species is different from each other those are the *S. vulgaris* has oval shaped buccal capsule, the *S. edentatus* has normal buccal capsule and the *S. equinus* has oval in outline buccal capsule [4].

Epidemiology: Large strongyles are a common parasite of horse throughout the world and causes deaths when control measures are neglected. In area with cold winters and mild summer, egg deposition peaks in spring and remains high over summer. At this time temperate is suitable for larval development and massive contamination of pasture. Infective larvae may occur in the summer and early autumn, when young susceptible horses are present. If the summer is hot and dry, only a small proportion of strongyle eggs develop to larvae and these may be short lived, but continual re-infection keeps pasture contamination high [16]. In Ethiopian, *S. vulgaris* is very common and highest in equids in mid and high altitude areas where the rain fall is relatively high and follows as a bimodal pattern [6].

Age is important in regard to susceptibility to infection and disease and foals and yearlings are usually the most affected while adults over five years of age usually harbor more moderate numbers of worms. Any factors that reduce immunity in adult horses are also likely to increase their strongyle burdens: for example dietary problems and over workload [9]. Higher egg numbers per gram are recorded in young horse (= 3 year) as compared with older horse. The excretion of eggs is not affected by the sex of the animals [17].

The main sources of parasites are horses infected with adult worms. Young animals of less than 4-5 years are usually the most heavily infected and, because of their greater susceptibility, adult female worms in the intestine of these animals are fairly prolific and excrete considerable numbers of egg in the feces. Adult horses tend to have lower parasite burdens because of the development of immunity; however, this resistance or immunity is incomplete, so the animals continue to pass low number of eggs in their feces, these contributing to significant contamination of pasture [9]. Strongylosis may occur in adult animals kept in sub-urban paddocks and subjects to overcrowding and poor management [3].

Life Cycle: The large strongyles have direct life cycle. Egg lay by the adult female worms, are passed in the feces to the external environment. Development from egg to L3 under good conditions requires approximately two weeks. Five developmental stages are recognized in the life cycle of these parasites: first larval stage (L1), second larval stage (L2), third larval stage (L3), fourth larval stage (L4) and fifth larval stage (L5). Infection of host animals occurs by ingestion of (L3). Subsequent parasitic larval development of the three species differs [3].

***S. vulgaris*:** The L3 penetrates the intestinal mucosa and molt to L4 in the sub mucosa. The larva enters to small arteries and migrates on the endothelium to their predilection site in the cranial mesenteric artery and its main branches. After a period of development of several months the larvae molts to L5 and return to the intestinal wall via arterial Lumina. Nodules are formed around the larvae mainly in the wall of the caecum and colon. The larvae does not travel further within the arteries due to their size and the subsequent rupture of the nodules release the young adult parasites into the lumen of intestine. The pre-patent period is 6-7 months [3].

***S. edentatus*:** After penetration of the intestinal mucosa L3 travel to the portal system and reach the liver parenchyma within a few days. About two weeks later they molt to L4 and then further migration takes place in the liver and, by 6-8 weeks post infection, larvae can be found sub peritoneal around the hepato-renal ligament. The larvae then travel under the peritoneum to many sites with the predilection for the flanks and hepatic ligaments. The final molt occurs after four months and each L5 then migrate, still sub peritoneal, to the wall of the large intestine where large purulent nodule is formed, which subsequently ruptures with the release of the young adult parasite into the lumen. The pre-patent period is 10-12 months [3].

***S. equinus*:** This species has least larval migration when comparing from the other two large strongyle species. It appears that the L3 loss their sheaths while penetrating the wall of the caecum and ventral colon and within one week provoke the formation of the nodules in the muscular sub serosal layers of the intestine. The larvae molt to L4 occurs within these nodules and then the larvae travel across the peritoneal cavity to the liver where they migrate within the parenchyma for six weeks or more. After this time L4 and L5 have been found in and around the pancreas before their appearance in the large intestinal lumen. The pre-patent period is 8-9 months [3].

In general the worm lays egg in the intestine, which passes out with the feces and hatch on the ground. The first and second larval stages are free living organisms. The third stage, fourth stage and fifth stages are parasitic stage. The third stage larva can infect the equids. They migrate onto the blades of the grass that the equids eats. At this point, the life cycle of the various strongyles begin to differ. Once swallowed the larva of the large strongyle molt in the intestine and then invade the intestinal wall. The larvae eventually come back out into the intestine and become adult [18].

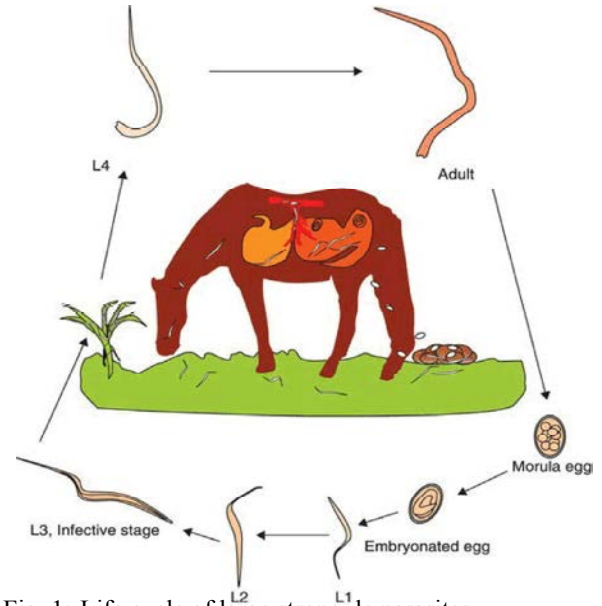

Fig. 1: Life cycle of large strongyle parasites
Source: [19].

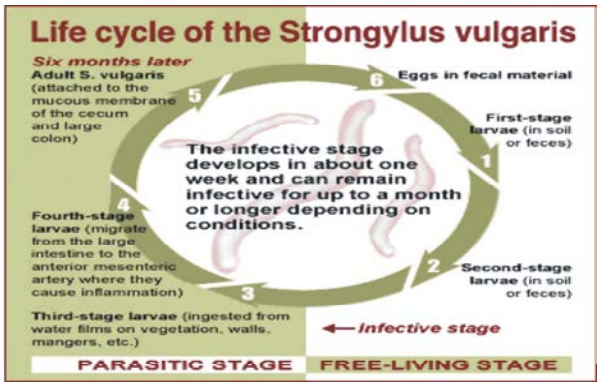

Fig. 2: Life cycle of *strongyle vulgaris*
Source: [20].

Pathogenesis of Large Strongyles: The disease process associated with the strongyle can be divided into those producing by migrating larvae and those producing by adult worms. Heavy intestinal infection can alter intestinal motility, permeability and absorption [3].

Larvae: The larval stages of the large strongyles have little effect but the *S. vulgaris* has a great effect in the arterial system of the intestinal tract of horses. Lesions are most common in the cranial mesenteric artery and its main branches and consist of thrombus formation provoked by larval damage to the endothelium together with a marked inflammation and thickening of the arterial wall. True aneurysms with dilatation and thinning of the arterial wall, although uncommon, may be found especially in animals which have experienced repeated infection [3].

Most of the information concerning on the *S. vulgaris* due to experimental infection of foals. A few weeks after infection with several hundred L3 a clinical syndrome of fever, inappetence and dullness occurs sometimes accompanied by colic. At necropsy, these signs are associated with arteritis and thrombosis of intestinal blood vessels with subsequent infarction necrosis of area of bowel, but the syndrome of this severity is not reported in foals under natural conditions, probably because larval intakes is continuous during grazing [3].

In *S. edentatus* infection-there are gross changes in the liver associated with early larval migration, but these rarely result in clinical signs. Similarly, the hemorrhages and fluid filled nodules which accompany later larval development in sub peritoneal tissues rarely result in clinical signs [3].The larvae of *strongylus* species returning to the intestine cause to large nodules in the wall of caecum and colon with a considerable hemorrhage may follow when these rupture and release the worm into lumen of the intestine. In the heavy burdens bleeding can occur and leads to the death of the animals [16].

Adults: The adult strongyles can be divided into those that cause blood losses and those that are superficial tissue damages. Strongyle species have large buccal cavity which they use to draw in and digest plugs of mucosa, while secreting anticoagulants to aid the ingestion of blood. Hemorrhage continuous from feeding points after worms detach to find a new attachment sites. *S. edentatus* and *S. equinus* adults are more harmful to the horse and suck more blood than *S. vulgaris* adults, but the larvae are not as dangerous [16].

The pathogenesis of infection with large strongyle species are associated with damage to large intestinal mucosa due to the feeding habit of the worms and, to some extent, to the disruption caused by emergence of young adults into the intestine following completion of their parasitic larval development. The effect of this parasite damage the blood vessels can causes to hemorrhage and ulcer [3]. Thrombo-embolism due to detached fragments of thrombus lodged in smaller vessels of the caecum and colon, ischemic or hemorrhagic infarction of intestinal wall and peritonitis and colic of varying severity [15].

Clinical Signs: In natural infestations it is often impossible to quantify the effect of individual strongyle species as a clinical picture usually represents the combined effects of a mixed infestation. Unthrifitness, poor hair coat, anemia if there is significant blood loss, impaired performance, poor appetite, depression, intestinal rapture, weight loss and death are signs associated with a wormy horse. The clinical signs of a large strongyle (primarily *S. vulgaris)* infection are directly associated with the larval migration in the walls of the mesenteric arteries. The inflammation constricts the arteries and reduced blood supply (ischemia) to the intestinal wall kills tissue (infarction). "Verminous aneurysm" or "worm aneurysm" is classic of this parasite [16].

The significance of migrating larvae of *S. vulgaris* in natural cases of colic is difficult to assess but it is generally recognized that where strongyle infection of horses are efficiently control the incidence of colic is markedly decrease. The frequency of colic is directly related to the parasite control program. More colic in untreated young horses will mean more in the adults. Horses will have intermittent diarrhea and lameness from emboli in the legs may appear with exercise and disappear with rest [3].

Pathology: *S. equinus* and *S. edentatus* causes to hemorrhagic tracts may be produced in the hepatic parenchyma from migrating larvae and parenchymal scars of fibrous tissue on the hepatic capsule are often found in postmortem. Migrating larvae may also elicit sub peritoneal hematoma, hemorrhage, peritonitis and omental adhesions. In the gut wall they may form nodules and hemorrhagic foci. The larvae localized in the cecal or portal veins and causes to perivascular thickening. The liver becomes swollen, looks bluish-red and has the white larvae embedded under its capsule [21].

The pathology of *S. vulgaris* lesions due to migrating larvae are most common in the cranial mesenteric artery and its main branches and consists of thrombus formation provoked by larval damage to the endothelium, together with a marked inflammation and thickening of the arterial wall. True aneurysms with dilatation and thinning of the arterial wall, although uncommon, may be found especially in animals which have experienced repeated infection [21].

Diagnosis: Diagnosis is based on the grazing history and clinical signs of loss of condition and anemia. Although oval, thin shelled strongyle eggs on fecal examination may be a useful aid to diagnosis. It is important to remember that substantial worm burdens may be associated with fecal egg counts of only a few hundred EPG due to either low fecundity of adult worms or to the presence of many immature parasites [3]. Diagnosis of the large strongyle infections can be assumed if eggs are

found in the feces. However, because of the prolonged pre patent period of all species in this group, eggs will not be present in the feces in animals less than 9-12 months old [22].

The most common finding in equine samples is the strongyloide egg that is oval shape. Equids are infected with the three large strongyle species, although individual species cannot be determined. Diagnosis based on the general physical condition, clinical sign, history, fecal egg count, microscopic examinations and unique husbandry or environmental circumstances [23].

Generally the diagnosis of large strongyle by: examination of feces for the detection of the strongyle egg, fecal flotation examination, but this test does not differentiate between large and small strongyles; fecal culture for identification of strongyle larvae and per rectal examination reveals aneurysm of cranial mesenteric artery [4]. In general a specific diagnosis is difficult to achieve in every case. Few clinical observations or laboratory results are pathognomonic for the disease syndrome associated with strongyle infection [16].

Treatment: Treatment may be targeted against immature and adult large strongyle worms in the lumen of the intestine, against migrating strongyle larvae, particularly *S. vulgaris* [16]. Usually, equines are treated with anthelmintic drugs to eliminate adult strongyles from the large intestines to prevent excessive contamination of pastures with eggs and L3. Some deworming treatments are less effective because the encysted larvae remain protected within a barrier wall for up to two and a half years. Thiabendazole has been widely used and several other drugs have been developed or approved for use in adult horses, including benzimidazole, tetrahydropyrimidines and organic phosphorus compounds [14].

There are a number of broad spectrum anthelmintics including the benzimidazole, pyrantel and ivermectins which are important in the removing lumen dwelling adult and larval strongyles and these are usually marketed as in-feed or oral preparation. Some modern benzimidazoles and the ivermectins are also efficient against developing stages of the migrating large strongyles [3].

Control and Prevention: The approach of most equine strongyle control programs goes into the category of prevention of pasture contamination that is prevention of excessive pasture contamination thereby minimizing the risk of exposure to infective larvae [24]. The mixed or alternative grazing with ruminants can reduce pasture infectivity as horse strongyles will not establish in these hosts. Removal of all horse feces from the fields twice weekly is highly effective for the control of strongyles [16].

Since horse of any age can be become infected on the excreted eggs, all grazing animals over two months of age should be treated every 4-8 weeks with effective broad spectrum anthelmintics. Any new animals joining a treated group must receive anthelmintic and be isolated for 48-72 hours before being introduced [3].

Key elements of an effective control program include strategic treatment and improve pasture management [15]. Regular treatments of all animals in any group of horses, starting from the weaners are typically used to eliminate adult strongyles and these prevent excessive contamination of pasture with eggs and infective larvae (L3).The rotation of dewormers has been suggested as a means of helping prevent parasites' developing resistance medications. The separation of different age group at grazing is recommended for the eradication of Strongylosis. The goal of strongyle control plan is to keep the number of adult, egg-bearing strongyles at low subclinical levels through the strategic use of selected, approved anthelmintics and programs that limit the horse's exposure to infected manure [9].

CONCLUSION

Equines are important animals for the life system of developing countries especially in Africa. These animals particularly used for transportation system. Equines provide unlimited services for man, so the equines are the subject of routine frequent neglect and maltreatment. Strongyle infection in equines is found to be widely prevalent and should be considered as one of the important disease of equines; particularly the large strongyles are the most important parasites of equines and more common in untreated equines and exerting a significant economic impact when they are raised. The large strongyle infections are also common in contaminated environments. These parasites will continue to be the most damaging parasite helminthes in the developing country. Based on the above conclusions the following recommendations are forwarded:

- Overworking should be minimized.
- Continues education must be given to owners about proper management of equines.
- Strategic treatment and improved pasture management must be practiced to prevent excessive pasture contamination.

- Any new animals must be isolated from a treated group for 48-72hours before being introduced to the group and must be receive anthelmintics.
- Equines must be kept separately in relation to housing and grazing.
- It is advised that there should be equine health promotion program supported by government.

REFERENCES

1. Addissalem, H., 2005. Health welfare assessment working donkey and mules in two zones of SNNP: Sidama and Wolayta zones, DVM thesis, Addis Ababa University, faculty of veterinary medicine, Debrezeit, Ethiopia.
2. Yilma, J.M., G. Feseha, E.D. Suendsen and A. Mohammed, 1991. Health problems of working donkey in Debrezeit and Menagesha region of Ethiopia. In Fielding and Pearson: donkey, mule and horse in tropical agricultural development university of Edin burgh, center for tropical veterinary medicine. UK, pp: 151-155.
3. Urquhart, G.M., J. Armour, J.I. Duncan, A.M. Dunn and F.W. Jennings, 1996. Veterinary Parasitology. 2nd ed. Blackwell Science Ltd., Oxford, pp: 42-47.
4. Mandal, S.C., 2012. Veterinary Parasitology at a Glance. 2nd ed. Revised and enlarged edition (Based on new VCL syllabus), pp: 230-236.
5. Nielsen, M.K., J. Monard and S.N. Olsen, 2006. Prescription only anthelmintics- a questionnaire surveillance and control of equine Strongyles in Denmark. Veterinary Parasitology, 135: 47-55.
6. Feseha, G., A. Mohammed and J.M. Yilma, 1991. Vermicular endoparasitism in donkey of Debrezeit and Menagesha, Ethiopia: Donkey, mule and horse in tropical agricultural development. Proc. Colloq on donkey, mule and horse. Eds. Fielding, D. and R.A. Pearson, University of Edinburgh, center for tropical veterinary medicine: UK, pp: 156-166.
7. Clarie Nichol, I. and W.J. Masterson, 1987. Molecular and biochemical characterization of surface antigen of *Strongylus vulgaris* of potential immunodiagnostic importance. Parasitology, 25: 29-38.
8. Tolliver, S.L., T. Lyons and J.H. Drudge, 1987. Prevalence of internal parasite in horse in critical tests of activity of parasiticides over 28-year period (1956-1983) in Kentucky. Veterinary Parasitology, 23: 273-284.
9. Lefevre, P.C., J. Blancou, R. Chermette and G. Ulienberg, 2010. Infectious and parasitic disease of livestock. French, 2: 1530-1544.
10. Owend, J. and D. Slocombe, 1985. Pathogenesis of helminthes in equines. Veterinary Parasitology, 18: 139-153.
11. Patton, S. and J.H. Drudge, 1977. Clinical response of pony foals experimentally infected with *Strongylus svulgaris*. American Journal of Veterinary Research, 38: 2059-2066.
12. Duncan, J.L. and H.M. Pirie, 1985. The pathogenesis of single experimental infections with *Strongylus vulgaris* in foals. Research in Veterinary Science, 18: 82-93.
13. Marinkovic, D., A.K. Sanja, V. Krstic and K. Milijana, 2009. Morphological findings in the cranial mesenteric artery of horses with verminous arteritis. Acta Veteterinaria, 59: 231-241.
14. Drudge, J.H., E.T. Lyons and S.C. Tolliver, 1975. Critical tests of suspension paste and pellet formulations of mebendazole in the horse. American Journal of Veterinary Research, 36: 435-439.
15. Kassai, T., 1999. Veterinary helmintology. 1st ed. London Real educational and professional publishing limited, pp: 60-63.
16. Radostits, O.M., C.C. Gay, K.W. Hinchcliff and P.O. Constables, 2007. Veterinary medicine, a text book of the disease of cattle, horse, sheep, pig and goats. 10th ed. London, pp: 1558-1562.
17. Saeed, K., Z. Qudir, K. Ashraf and N. Ahmad, 2010. Role of intrinsic and extrinsic epidemiological factors on strongylosis in horses. Journal of Animal and Public Science, 20(4): 277-280.
18. Shaprio, L.S., 2004. Pathology and Parasitology for veterinary technicians, pp: 183-184.
19. Reinemeyer, C.R. and M.K. Nielsen, 2012. handbook of equine parasite control. Blackwell publishing, pp: 45-60.
20. Extension, 2014. Available on http://www.extension.org/pages/10280/strongyles-in-horses (Accessed on May 10, 2015).
21. Taylor, M.A., R.L. Coop and R.L. Wall, 2007. Veterinary Parasitology. 3rd ed. Blackwell science, pp: 280-284.
22. Rose, R.F. and D.R. Hodgson, 1999. Manual of equine practice. 2nd ed. London, pp: 330-331.
23. Soulsby, E.J.L., 1986. Helminthes, arthropods and protozoa of domesticated animals. 7th ed. London: The English language book society, Baillier Tindal, pp: 650-700.
24. Marquartd, C., S. Riochard, D. Robert and B. Grieve, 2000. Parasitology and vector biology. 2nded. Canada Harcourt Academic press, pp: 381-385.